LETTER TO THE EDITOR EDİTÖRE MEKTUP

Burkitt Lymphoma with Severe Lactic Acidosis

Ağır Laktik Asidozlu Burkitt Lenfoma

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Dear Editor,

The prognosis of Burkitt lymphoma mainly depends on histopathology, the degree of involvement, and the characteristics of the patient (1). Reports indicating that lactic acidosis in patients diagnosed with malignancy is resolved only with chemotherapy are rare. Burkitt's lymphoma is the fastest growing human tumor, with a cell doubling time of 24–48 hours, so treatment should be started quickly without delay (2). There is a significant risk of tumor lysis syndrome when treating Burkitt lymphoma (3).

An 11-year-old boy who was followed up for autism with no other known disease was investigated in Iraq with a preliminary diagnosis of pancreatitis, with complaints of abdominal pain and vomiting that started 2 weeks prior. Hepatosplenomegaly and lymphadenopathy were not detected. However, fever, abdominal pain, vomiting, and positive inflammatory markers were observed. Mitral regurgitation was detected on the echocardiogram, and adrenaline infusions of 0.1 µg/kg/min and 0.5 µg/kg/minute milrinone were started.

The laboratory findings revealed a mild increase in C-reactive protein at 14.6 µm g/L. The initial complete blood count revealed cytopenia with anaemia (7.8 g/dL) and thrombocytopenia (49×10^9/L) without blast cells in the peripheral smear. Immunoglobulin and albumin levels were low. Triglyceride, D-dimer, ferritin, and lactate dehydrogenase levels were high. Troponin-T, BNP, amylase, and lipase levels were normal. Thyroid ultrasound revealed a hypoechoic lesion in the left thyroid lobe. Abdominal computed tomography revealed hypoechoic lesions 20×10 mm and 11×8 mm in size in the pancreas and diffuse soft tissue densities around the mesentery and superior mesenteric artery vein. When bone marrow aspiration was performed on the patient, 90% blasts were detected, and Burkitt lymphoma was diagnosed (Figure 1). The patient was evaluated as having stage 4 disease according to the Ann Arbor Staging System. The first curative chemotherapy with cyclophosphamide, vincristine, and steroid chemotherapy plan was made according to the Child Oncology Group ANHL01P1 Protocol. Electrolytes were checked at frequent intervals. Aggressive fluid hydration, allopurinol, and rasburicase treatment were used to prevent tumor lysis syndrome.

Keywords
Burkitt lymphoma, lactic acidosis, hypoglycemia

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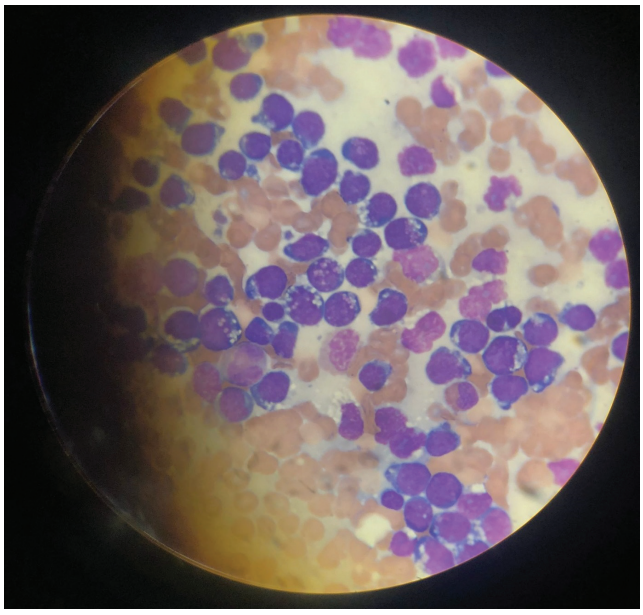

Figure 1. The patient's bone marrow aspiration image (bone marrow aspirate and biopsy slides) revealed complete effacement of the normal bone marrow architecture by sheets of monomorphic-looking cells. At high-power magnification, the medium-large neoplastic cells had a FAB L3 morphology, round or oval nuclei, finely stippled nuclear chromatin, multiple nucleoli and a thin rim of deeply basophilic cytoplasm containing numerous prominent vacuoles. Hematoxylin-eosin stain)

After the patient was admitted to the intensive care unit, the lactate levels in the blood gases first increased and then decreased with chemotherapy (6.7–8.9–13.7–12.2–16–14.3–10.2–6.9–15–20–20–20–23–25–25–23–20–15–10–8.4–4–2.6 mmol/L). When the patient's lactate level was 25 mmol/L, the patient's lactate level rapidly decreased after chemotherapy started. The lactate level decreased to 2.6 mmol/L 48 hours after chemotherapy started.

One of the most unusual features of our patient was severe lactate elevation and accompanying hypoglycemia. There are rare cases of lactic acidosis and hypoglycemia in non-Hodgkin lymphomas in the literature (4,5). Cancer cells can continue glycolysis even in the presence of high oxygen and therefore produce high levels of lactate. Insulin-like growth factors and their receptors, which are overexpressed by some cancer cells, can mimic many of the activities of insulin. A review of the literature revealed that lactic acidosis in patients diagnosed with malignancy is resolved only with chemotherapy (5).

Literature-Based Pathophysiological Mechanisms

The pathophysiology of type B lactic acidosis in pediatric malignancies is multifactorial. The central mechanism is tumor-driven metabolic reprogramming, classically described as the Warburg effect. In this process, malignant cells preferentially utilize aerobic glycolysis over oxidative phosphorylation even in the presence of adequate oxygen, leading to excessive lactate production. This is further amplified by the upregulation of key enzymes, including pyruvate kinase M2, lactate dehydrogenase A, and pyruvate dehydrogenase kinase 1, which accelerate glycolytic flux toward lactate accumulation (6).

In addition, impaired clearance exacerbates systemic acidosis. Malignant infiltration of the liver and kidneys significantly reduces lactate metabolism and excretion (6,7). Thiamine deficiency, often overlooked in critically ill oncology patients receiving inadequate nutrition or prolonged parenteral support, further impairs pyruvate oxidation by blocking its conversion to acetyl-CoA. Importantly, supplementation with thiamine has been shown to result in rapid reversal of severe lactic acidosis in such contexts (8). Taken together, these mechanisms underscore the multifactorial nature of malignancy-associated lactic acidosis, highlighting both tumor-intrinsic metabolic alterations and reversible cofactors that must be addressed to optimize patient outcomes.

In conclusion, lactic acidosis is frequently observed in circulatory failure and hypoxemia in critically ill children, but it rarely occurs for different causes, such as in our patient. Lactic acidosis in patients diagnosed with malignancy can be resolved only with chemotherapy.

Footnotes

Conflict of Interest: No conflict of interest was declared by the authors.

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